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Concise Oxford Textbook of Medicine, 1st Edition

Chapter 13.3

The motor and sensory systems, midbrain, and brainstem

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The motor system

The lower motor neurone

The motor unit consists of a number of muscle fibres, ranging from 100 in the facial muscles to 2000 in the quadriceps, supplied by a single, fast-conducting, α-motor fibre. These originate from large cells

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in the anterior horns of the grey matter of the spinal cord and in the somatic motor nuclei of the cranial nerves, and are known as the lower motor neurones. Loss of function of the anterior horn cells, or interruption of their axons, causes weakness or paralysis of the muscles they supply, with loss of stretch reflexes, shown by flaccidity and loss of tendon jerks, and, if paralysis persists, wasting of the muscle due to loss of excitable tissue.

The upper motor neurone

The main descending motor pathway is derived from neurones in the precentral gyrus. Movements of the foot are controlled by neurones on the medial surface of the hemisphere, and on the lateral surface from above downwards are the areas for the leg, trunk, arm, hand, face, and tongue, although these are not rigidly demarcated centres. Descending axons converge in the posterior limb of the internal capsule and then occupy the middle third of the cerebral peduncle. In the pons the fibres are more dispersed and become concentrated again in the prominent pyramid, from which the name of the pyramidal tract is derived, on the anterior surface of the medulla. Most of the fibres cross at the lower end of the medulla and descend in the lateral columns of the spinal cord. Only a small proportion of the corticospinal fibres synapse directly on lower motor neurones and most terminate on interneurones.

An acute complete lesion of the corticospinal tract causes flaccid paralysis with loss of tendon reflexes. With the passage of time or with a partial or progressive lesion the characteristic effect is spastic weakness or paralysis. Loss of power is accompanied by an increase in stretch-reflex activity. In the upper limb the weakness is usually most evident in distal muscles, but movements of the hip joint may be affected when no other loss of power can be found in the lower limb. Loss of fine movements, particularly of the fingers, is often much more prominent than loss of strength. In many complex movements, such as walking,

the normal precise sequence of contraction and relaxation of opposing muscles is lost.

The increased resistance to passive movement is more marked in the flexor muscles of the arm and the extensor muscles of the leg. It often has a ‘clasp knife’ character, in that the resistance suddenly lapses due to reflex lengthening. The tendon reflexes are exaggerated and reflex contractions can often be elicited in muscles other than those usually examined.

The normal plantar reflex consists of flexion of all the digits on firm stroking of the lateral side of the sole. In the extensor, or Babinski, reflex the big toe dorsiflexes and the other toes fan. This response is reliably found when an upper motor-neurone lesion or loss of function is undeniably present, but is far less useful in cases of doubt, when it is often recorded as ‘equivocal’.

A curious feature of upper motor-neurone lesions is loss of certain cutaneous reflexes. The superficial abdominal reflexes, elicited by stroking the skin in each quadrant, are the best known, although a variable, finding, particularly after middle age, but unilateral loss is occasionally a valuable sign.

In the distribution of the cranial nerves an acute upper motor-neurone lesion usually causes dysphagia and dysarthria for a few days only. Bilateral lesions cause persistent symptoms, with slow tongue movements and often an exaggerated jaw jerk, elicited by a tap on the chin with the mouth half open. This condition is known as pseudobulbar palsy (the ‘bulb’ being the medulla) to distinguish it from the lower motor-neurone bulbar palsy. It is often accompanied by mild limb spasticity, a shuffling gait, and great lability of emotional expression.

An upper motor-neurone facial-nerve palsy differs in a number of respects from a nuclear or peripheral palsy. The muscles of the upper face are relatively or entirely spared, apparently because of bilateral cortical control.

The sensory system

The afferent inflow from skin, muscles, tendons, and joints arises from end-organs specifically adapted to respond to appropriate stimuli and also from a non-specific network of cutaneous nerve endings. In the clinical context it is the anatomy of the sensory pathways that is of obvious relevance.

The afferent fibres from the limbs are formed by one branch of the axons of the neurones of the posterior root ganglia, the other branch of which enters the spinal cord. The posterior spinal-root fibres enter the grey matter where many, apparently concerned with reflex activity, form synapses with interneurones or anterior horn cells. The main afferent stream divides: axons concerned mainly with postural sense and with some aspects of touch, and probably with vibration sense, proceed in the posterior columns of the same side of the spinal cord to the dorsal-column nuclei in the medulla, from which arise the secondary sensory axons, which decussate and ascend in the medial lemniscus to the thalamus. Fibres concerned with pain and thermal sensation synapse in the posterior horns and the axons of the secondary relay pass upwards for a few segments on the same side before decussating in the centre of the cord and passing up in the lateral columns as the spinothalamic tracts. These lie laterally in the medulla but eventually join the medial

lemniscus and enter the thalamus. The sensory relay is continued to the postcentral gyrus and to a wide area of the posterior part of the cerebral hemisphere. Many afferent fibres convey information that does not enter consciousness but is concerned with reflex activity or with the afferent flow to the cerebellum.

Sensory loss from interruption of a peripheral nerve or posterior root affects all modalities. However, cutting a single posterior root may result in no detectable sensory loss, because of overlap from neighbouring roots. Similarly, the area of sensory loss resulting from a peripheral nerve lesion will be much less extensive than the full distribution of the nerve. Within the central nervous system the separation of the sensory tracts in the spinal cord allows selective loss of different sensory modalities. Pain and thermal sense will be impaired when the spinothalamic tracts are damaged, while lesions of the posterior columns result in loss of postural sense. Sense of touch is distributed between both pathways, the element involved in tickle passing through the spinothalamic tracts.

Lesions of the parietal cortex may result in loss of all forms of sensation, including that of pain, but sometimes there is severe loss of discriminatory forms of sensation with retention of appreciation of cruder modalities.

Symptoms

Patients' complaints arising from disorders of the sensory system are often difficult to interpret. Loss of cutaneous sensation may be sufficiently obvious, as with the complaint of being unable to feel the feet on the floor or to judge the temperature of the bath water, but other sensory symptoms are less easy to attribute to disturbance of a particular modality and the distinction of positive from negative

symptoms is also difficult. The familiar paraesthesias, ‘pins and needles’, may apparently result from lesions of the sensory pathways at any level. A sensation as of a tight bandage round the leg is commonly complained of by patients with a lesion of the posterior columns of the spinal cord.

Loss of proprioceptive sensation results in sensory ataxia. Difficulty in maintaining balance is greatly increased when information derived from vision is also lost, leading, on examination, to Romberg's sign, consisting of falling when standing with feet together and eyes closed, and to the complaint of being unable to walk outside after dusk.

Pain may result from disease of the peripheral and, less commonly, of the central nervous system. Compression of a peripheral nerve, or more particularly of a dorsal root, may cause paraesthesias and pain in the distribution of the sensory fibres. Pain of a peculiarly distressing, burning character can arise from lesions of the spinothalamic tract in the spinal cord and brainstem, and similar and even more persistent pain and dysaesthesia, or unpleasantly altered cutaneous sensation on stimulation, from thalamic lesions.

Subcortical lesions: internal capsule, midbrain, and brainstem

The internal capsule

In the internal capsule the descending motor fibres are condensed into a small space immediately anterior to the similarly narrowly localized ascending fibres. Even relatively small lesions in this area can therefore cause severe hemiplegia of the opposite limbs, the degree of sensory loss depending on the extent of the lesion.

The midbrain

Among the crowded tracts and nuclei of the midbrain those that can most readily be identified as contributing to the symptomatology of lesions in this area are the descending corticospinal and corticobulbar tracts, the nuclei of the third and fourth cranial nerves, the reticular formation, and, more speculatively, the red nucleus. The contiguous superior cerebellar peduncles may also be involved.

Weber's syndrome of a third-nerve palsy and crossed hemiplegia is the result of a lesion of the cerebral peduncle involving the third nerve as it leaves the brain. Benedikt's syndrome of a third-nerve palsy with involuntary movements of the opposite limbs is thought to result from a lesion of the red nucleus.

A characteristic sign of involvement of the upper midbrain is Parinaud's syndrome, which, in its complete form, consists of paralysis of vertical gaze and of convergence.

Lesions involving the reticular formation have been held responsible for disturbances of conscious level and also for the condition of akinetic mutism in which the patient makes no voluntary movement except of the eyes.

The pons and medulla

The pons and medulla contain nuclei of the fifth to the twelfth cranial nerves, important cerebellar connections, and motor, sensory, and autonomic pathways.

The lateral medullary syndrome of Wallenberg is relatively common. It includes dysphagia and dysarthria (ninth and tenth nerve nuclei), vomiting and hiccup (nucleus ambiguus), and vertigo (vestibular nuclei) combined with cerebellar ataxia of the limbs on the side of the lesion (inferior cerebellar peduncle), ipsilateral Horner's syndrome (descending autonomic fibres), loss of pain and thermal sensation on the face on the side of the lesion (fifth nerve nucleus) and in the opposite limbs (lateral lemniscus). There is no weakness as the pyramidal tracts are spared.

The rare medial medullary syndrome consists of weakness and loss of postural sense in the limbs on the side opposite to the lesion (pyramidal tract and medial lemniscus) and ipsilateral paralysis of the tongue (twelfth nerve nucleus).

Certain signs of brainstem pathology may be encountered in isolation or combined with evidence of widespread disease. An internuclear ophthalmoplegia of the form commonly seen consists of failure of adduction of the eye in conjugate gaze to one or both sides, but with preservation of convergence, indicating that the medial rectus is not paralysed but cannot act in conjunction with the opposite lateral rectus. This results from a lesion of the medial longitudinal bundle connecting the nuclei of the third and sixth nerves.

The locked-in syndrome results from interruption of the descending and ascending long

tracts in the brainstem. As no speech or movement of the limbs is possible, it is easy to assume that consciousness is lost, but this is not so and such patients readily learn to communicate by using eye-movement signals. This syndrome differs from akinetic mutism where, although the eyes are moved, no communication is possible.

Further reading

Bickerstaff, E.R. and Spillane, J.A. (1989). *Neurological examination in clinical practice*, 5th edn. Blackwell Scientific, Oxford.

Brodal, A. (1981). *Neurological anatomy in relation to clinical medicine*, 3rd edn. Oxford University Press.